Received: 4 May 2021 | Revised: 12 August 2021 | Accepted: 24 August 2021

DOI: 10.1111/dom.14533

BRIEF REPORT

WILEY

Glycaemic efficacy of an expanded dose range of dulaglutide according to baseline glycated haemoglobin (HbA1c) subgroup: Post hoc analysis of AWARD-11

Juan P. Frias MD[1] | Enzo Bonora MD[2] | David A. Cox PhD[3] | M. Angelyn Bethel MD[3] | Anita Y.M. Kwan MSc[3] | Sohini Raha PhD[3] | Raleigh E. Malik PhD[3]

[1]National Research Institute, Los Angeles, California, USA

[2]University and University Hospital of Verona, Verona, Italy

[3]Eli Lilly and Company, Indianapolis, Indiana, USA

Correspondence
Dr. Raleigh E. Malik, PhD, Eli Lilly and Company, Lilly Corporate Center, Indianapolis, IN 46285, USA.
Email: rmalik@lilly.com

Funding information
Eli Lilly and Company

Abstract

The AWARD-11 trial demonstrated the safety and efficacy of dulaglutide 3.0 and 4.5 mg compared to dulaglutide 1.5 mg in patients with type 2 diabetes inadequately controlled with metformin. This post hoc analysis examined the change from baseline in glycated haemoglobin (HbA1c) and proportions of patients achieving HbA1c <7% at weeks 36 and 52 with dulaglutide 1.5 mg, 3.0 mg or 4.5 mg across clinically relevant baseline HbA1c subgroups (<8%; 8.0% to < 9.0%; 9.0% to < 10%; and ≥ 10%). Mean reductions in HbA1c were observed across all baseline HbA1c subgroups at 36 weeks (range of HbA1c change: 1.5 mg: −1.0% to −2.2%; 3.0 mg: −1.2% to −2.5%; and 4.5 mg: −1.2% to −3.2%). More patients randomized to 3.0 mg or 4.5 mg (vs. 1.5 mg) achieved HbA1c <7% at 36 weeks regardless of baseline HbA1c; the difference in proportions was greater at higher baseline HbA1c (*P*-interaction = 0.096). Similar patterns in glycaemic improvement and proportions achieving HbA1c <7% were observed at 52 weeks. Hypoglycaemia and gastrointestinal adverse events were similar among the HbA1c subgroups. Glycaemic control was improved with dulaglutide dose escalation from 1.5 mg to 3.0 mg or 4.5 mg across baseline HbA1c subgroups (<8%; 8.0% to < 9.0%; 9.0% to < 10%; and ≥ 10%).

KEYWORDS
clinical trial, dulaglutide, GLP-1, glycaemic control, type 2 diabetes

1 | INTRODUCTION

In type 2 diabetes, sustaining glycaemic control over time often requires treatment intensification via dose escalation of ongoing therapy or initiation of new therapy (add-on or switch).[1] Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are recommended for many patients for glycated haemoglobin (HbA1c) reduction and cardiovascular benefit without increased risk of hypoglycaemia or weight gain.[2] Real-world outcomes confirm glycaemic and weight benefits for GLP-1RAs compared to other oral agents or insulin.[3,4] Recently approved higher doses of the once-weekly GLP-1RA dulaglutide (3.0 and 4.5 mg) have provided options to intensify treatment if needed.[5]

The AWARD-11 (The Assessment of Weekly AdministRation of LY2189265 in Diabetes-11) trial demonstrated that, in patients with

type 2 diabetes inadequately controlled with metformin monotherapy, escalation from dulaglutide 1.5 mg to 3.0 mg or 4.5 mg provided clinically relevant, dose-related improvements in glycaemic control with a similar safety profile.[6] HbA1c reductions from baseline were generally dose-dependently greater with all three dulaglutide doses in patients with higher (≥8.5%) versus lower (<8.5%) HbA1c levels at baseline.[6] The present exploratory post hoc analysis of AWARD-11 provides a more detailed analysis to assess the effect of dulaglutide 3.0 and 4.5 mg versus 1.5 mg on HbA1c reduction and the proportion of patients achieving glycaemic control (HbA1c <53 mmol/mol) across a range of clinically relevant baseline HbA1c subgroups representing varying levels of glycaemic control. These data are of value for personalizing antihyperglycaemic therapy to individual patient needs, and for assessing which patients may benefit more from dulaglutide dose escalation.

2 | MATERIALS AND METHODS

2.1 | Study design and population

The study design of AWARD-11 was previously described in detail.[6] Briefly, patients in this randomized, phase 3, double-blind, multicentre, parallel-arm study (ClinicalTrials.gov Identifier: NCT03495102) initiated treatment with once-weekly dulaglutide 0.75 mg for 4 weeks, followed by stepwise dose escalation every 4 weeks to the randomized dose of 1.5, 3.0 or 4.5 mg (Figure S1). Key eligibility criteria included age ≥ 18 years, HbA1c ≥58 mmol/mol (7.5%) and ≤ 97 mmol/mol (11.0%), body mass index (BMI) ≥25 kg/m^2, and stable-dose (≥1500 mg daily) metformin treatment (Table S2).

2.2 | Efficacy measures and safety assessments

The primary efficacy measure (change in HbA1c from baseline) and secondary efficacy measures (proportion of patients achieving HbA1c <53 mmol/mol [7.0%]; change from baseline in fasting serum glucose level; and change from baseline in body weight) were previously reported.[6] For the present exploratory analysis, change in HbA1c from baseline and the proportion of patients achieving HbA1c <53 mmol/mol at 36 and 52 weeks were assessed in the following subgroups of decreasing glycaemic control defined by baseline HbA1c: <8%; 8% to < 9%; 9% to < 10%; and ≥10%.

United States health plans use the Healthcare Effectiveness Data and Information Set (HEDIS®) quality measure of HbA1c >9% as the threshold to define "poor glycaemic control".[7] Thus, the change in HbA1c from baseline at 36 and 52 weeks in this HEDIS-defined subgroup was evaluated in an additional analysis by baseline HbA1c subgroups of ≤9% and > 9% (Figure S3).

Safety assessments included incidence of common gastrointestinal (GI) events (nausea, vomiting, and diarrhoea) and occurrence of hypoglycaemic episodes (clinically significant hypoglycaemia [Level 2, ie, plasma glucose level < 3.0 mmol/L or 54 mg/dl]; severe hypoglycaemia [Level 3, ie, event characterized by altered mental and/or physical functioning requiring assistance from another person for recovery[8]) up to 52 weeks.

2.3 | Statistical analysis

The analysis population for both efficacy and safety included randomized patients who received ≥1 dose of study medication. Efficacy analyses included patients with a baseline and ≥ 1 post-dose measurement for the variable of interest, and excluded measurements collected after discontinuation of study drug or initiation of another antihyperglycaemic medication. Efficacy analyses were carried out at 36 and 52 weeks and safety analyses were carried out at 52 weeks.

A mixed model for repeated measures (MMRM) was implemented within the HbA1c subgroups for assessing the change in HbA1c. An MMRM with interaction term between the treatment and the HbA1c subgroups was performed for the change from baseline in HbA1c. A longitudinal logistic regression model within the HbA1c subgroups and another longitudinal logistic regression model with the interaction term between the treatment and the HbA1c subgroups were implemented to analyse the proportion of patients with HbA1c <53 mmol/mol. *P* values were calculated for the main effects at a significance level of 0.05, while relevant interactions between HbA1c subgroups and treatment were calculated using a significance level of 0.10.

3 | RESULTS

3.1 | Baseline demographics and patient characteristics

Selected demographic and patient characteristics at baseline among HbA1c subgroups are shown in Table S1. Overall, patients with higher baseline HbA1c tended to be younger and had a longer duration of diabetes, higher fasting glucose levels, and lower C-peptide levels at baseline ($P < 0.05$).

3.2 | Efficacy

Dulaglutide 1.5 mg reduced HbA1c across all baseline HbA1c subgroups at 36 weeks (range −1.0% to −2.2%), effects that were sustained throughout the 52 weeks (range −1.0% to −2.1%; Figure 1A, C). HbA1c reductions were greater in patients randomized to dulaglutide 3.0 or 4.5 mg versus 1.5 mg in each HbA1c subgroup, with greater dose-related improvements in patients with higher baseline HbA1c up to 36 weeks (for doses of 1.5 mg, 3.0 mg and 4.5 mg, respectively, least-squares mean changes were: HbA1c subgroup <8%: −1.0%, −1.2% and −1.2%; HbA1c subgroup 8.0% to < 9.0%: −1.4%, −1.6% and −1.8%; HbA1c subgroup 9.0% to < 10%: −2.1%, −2.2% and −2.3%; and HbA1c subgroup ≥10%: −2.2%, −2.5% and −3.2%; *P*-interaction <0.001 [Figure 1A]). Greater HbA1c reduction

with dose escalation in patients with higher baseline HbA1c was maintained up to 52 weeks (Figure 1C, Figure S2).

More patients randomized to 3.0 mg or 4.5 mg achieved HbA1c <53 mmol/mol versus those on 1.5 mg at 36 weeks, regardless of baseline HbA1c. However, the difference across dose groups was greater at higher baseline HbA1c, with over half of patients randomized to dulaglutide 4.5 mg achieving HbA1c <53 mmol/mol in every baseline HbA1c subgroup (*P*-interaction = 0.096; Figure 1B). Similar patterns in the proportion of patients at HbA1c <53 mmol/mol were maintained to 52 weeks (*P*-interaction = 0.03; Figure 1D). Subgroup analysis based on the HEDIS-defined subgroups also showed results consistent with those in Figures 1C, S2 and S3).

From the scatterplot of discrete categories of HbA1c, across a broad range of mean baseline HbA1c values (7.6%-10.4%), there was a near-linear relationship between baseline HbA1c and change in HbA1c in each dulaglutide dose group at Week 52 (Figure S4). The slopes from the regression analysis on individual patient data were −0.59 (95% confidence interval [CI] -0.68 to −0.5; $P < 0.001$), −0.60 (95% CI −0.68 to −0.52; $P < 0.001$) and −0.76 (95% CI −0.85 to −0.67; $P < 0.001$) for dulaglutide 1.5 mg, 3.0 mg and 4.5 mg, respectively. The adjusted R^2 from the regression analysis was 0.25, 0.30 and 0.37 for dulaglutide 1.5 mg, 3.0 mg and 4.5 mg, respectively, suggesting a good proportion of the variability in change in HbA1c can be explained by the baseline HbA1c, and the proportion is greater in higher dose groups. When comparing these slope values between the treatment groups, the difference between the 3.0-mg and 1.5-mg doses was not significant ($P = 0.802$); however, differences were significant between the 4.5-mg dose and both the 1.5-mg and 3.0-mg doses ($P = 0.049$ and 0.022, respectively).

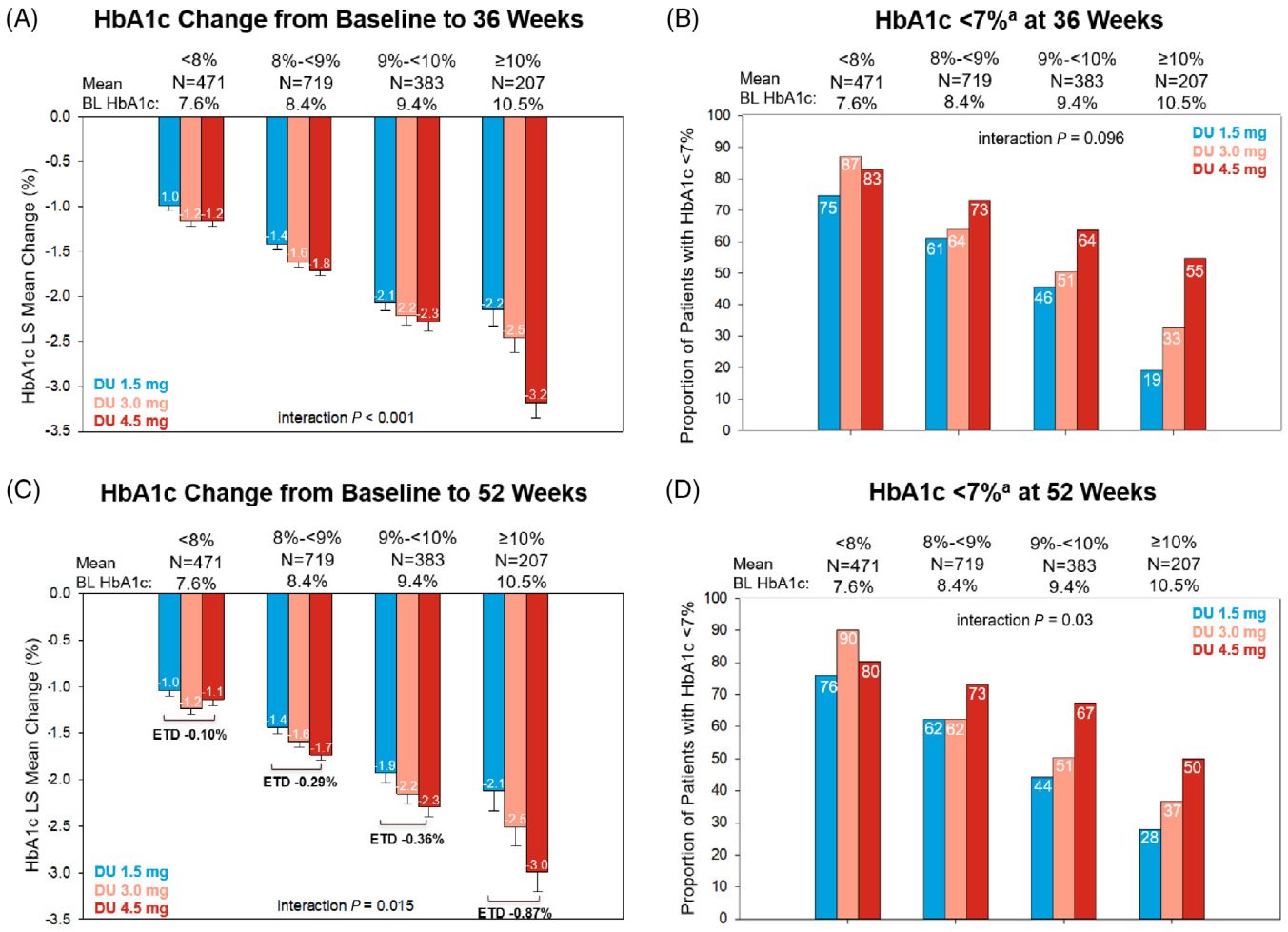

FIGURE 1 Glycated haemoglobin (HbA1c) change from baseline and proportion of patients at HbA1c target at weeks 36 and 52. N = patients with non-missing baseline value and at least one nonmissing post-baseline value of the response variable. Analyses included data while on treatment without additional antihyperglycaemic medication. A, Change in HbA1c from baseline to 36 weeks (primary time point; mixed model for repeated measures [MMRM]. B, Proportion of patients achieving HbA1c <53 mmol/mol at 36 weeks, longitudinal logistic regression. C, Change in HbA1c from baseline to 52 weeks, MMRM. D, Proportion of patients achieving HbA1c <53 mmol/mol at 52 weeks, longitudinal logistic regression. *P* values are for interaction of subgroup and treatment at Weeks 36 and 52, evaluated using a significance level of 0.10, unadjusted. [a]American Diabetes Association current guidelines for treating type 2 diabetes recommend an HbA1c of <53 mmol/mol (7%). BL, baseline; DU, dulaglutide; ETD, estimated treatment difference 4.5 mg versus 1.5 mg; HbA1c, glycated haemoglobin; LS, least-squares

3.3 | Safety

Consistent with the primary study population, the most frequent GI events experienced in all baseline HbA1c subgroups were nausea (range 13.6%-21.2%), diarrhoea (range 5.8%-16.7%) and vomiting (range 4.6%-13.6%). GI events increased with increasing doses, but the pattern was consistent regardless of baseline HbA1c. There was no statistically significant interaction for any of these GI events, suggesting that the effect of the dulaglutide dose on the occurrence of common GI events was not affected by baseline HbA1c (Table 1).

Incidence of documented hypoglycaemia (<3.0 mmol/L or <54 mg/dl) was low (range 0%-2%) and was not associated with baseline HbA1c across doses (Table 1). Two patients in the HbA1c <8% subgroup reported severe hypoglycaemia.

4 | DISCUSSION

The AWARD-11 trial demonstrated that dulaglutide 3.0 or 4.5 mg versus 1.5 mg once weekly provided dose-related improvements in glycaemic control at 36 weeks that were sustained up to 52 weeks.[6] This exploratory post hoc analysis suggests that glycaemic control was greater with dulaglutide 1.5 mg, 3.0 mg or 4.5 mg across subgroups of increasing HbA1c, with the higher doses resulting in greater improvements in glycaemic control, without increased incidence of hypoglycaemia. GI events increased with dulaglutide 3.0 or 4.5 mg, but the pattern did not differ across baseline HbA1c subgroups.

The largest HbA1c reductions were observed for those at highest baseline HbA1c regardless of dulaglutide dose, consistent with previous studies of glucose-lowering medications.[9-12] Moreover, the additional HbA1c-lowering achieved with escalation to 3.0 mg or 4.5 mg compared to 1.5 mg was increased with higher baseline HbA1c levels. For those initiating treatment in the highest baseline HbA1c subgroup (≥10%), escalation to dulaglutide 4.5 mg resulted in a mean HbA1c reduction of >3%, with the majority of patients achieving an HbA1c at or below the general recommendation of <53 mmol/mol.

For patients needing to intensify to injectable medications, guidelines recommend combination injectable therapy for patients with very poorly controlled glycaemia (defined as an HbA1c >10% or >2% above individualized target).[2,13] However, this analysis suggests that with the availability of a broader range of therapeutic doses, dulaglutide has the potential to bring a large proportion of patients with very poorly controlled glycaemia to clinical target without the need for addition of insulin. This approach has the advantage of not complicating polypharmacy and does not increase risk of hypoglycaemia or weight gain often associated with insulin.

Approximately 75% of patients with a baseline HbA1c <8% on dulaglutide 1.5 mg achieved HbA1c <53 mmol/mol, while

TABLE 1 Incidence of gastrointestinal and hypoglycaemic events by dulaglutide dose group and baseline glycated haemoglobin subgroup up to 52 weeks

			Baseline HbA1c subgroup				
		Overall (N = 1842)	<8% (N = 481)	8% to < 9% (N = 748)	9% to < 10% (N = 400)	≥10% (N = 213)	*P*-interaction
GI event							
Nausea	DU 1.5 mg	87 (14.2)	21 (13.7)	34 (14.0)	23 (15.2)	9 (13.6)	0.867
	DU 3.0 mg	99 (16.1)	29 (17.2)	40 (15.9)	18 (15.8)	12 (14.8)	
	DU 4.5 mg	106 (17.3)	33 (20.8)	37 (14.6)	22 (16.3)	14 (21.2)	
Vomiting	DU 1.5 mg	39 (6.4)	7 (4.6)	14 (5.8)	13 (8.6)	5 (7.6)	0.758
	DU 3.0 mg	56 (9.1)	17 (10.1)	22 (8.7)	8 (7.0)	9 (11.1)	
	DU 4.5 mg	62 (10.1)	18 (11.3)	22 (8.7)	13 (9.6)	9 (13.6)	
Diarrhoea	DU 1.5 mg	47 (7.7)	11 (7.2)	14 (5.8)	15 (9.9)	7 (10.6)	0.485
	DU 3.0 mg	74 (12.0)	26 (15.4)	25 (9.9)	10 (8.8)	13 (16.0)	
	DU 4.5 mg	71 (11.6)	15 (9.4)	26 (10.2)	19 (14.1)	11 (16.7)	
Hypoglycaemia							
Documented (<3.0 mmol/L or <54 mg/dl)	DU 1.5 mg	8 (1.3)	3 (2.0)	3 (1.2)	2 (1.3)	0 (0)	
	DU 3.0 mg	2 (0.3)	0 (0)	1 (0.4)	1 (0.9)	0 (0)	NA
	DU 4.5 mg	7 (1.1)	3 (1.9)	2 (0.8)	2 (1.5)	0 (0)	
Severe (excluding	DU 1.5 mg	1 (0.2)	1 (0.7)	0 (0)	0 (0)	0 (0)	
	DU 3.0 mg	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	NA
post-rescue)	DU 4.5 mg	1 (0.2)	1 (0.6)	0 (0)	0 (0)	0 (0)	

Note: Analyses based on all randomized patients who received at least one dose of study drug. Data presented as n (%). *P* values for interaction were not calculated for hypoglycaemia due to the small number of events. N = population size, n = number of patients with events.
Abbreviations: DU, dulaglutide; GI, gastrointestinal; HbA1c, glycated haemoglobin.

proportionally more patients (≥83%) with a baseline HbA1c <8% escalated to 3.0 mg or 4.5 mg achieved this target by 36 weeks. Although the increase in those achieving target was modest in this group, these findings suggest that escalation to dulaglutide 3.0 mg or 4.5 mg may be a viable option to achieve glycaemic targets for patients with moderately elevated HbA1c unable to achieve or maintain glycaemic control on lower dulaglutide doses. From a patient perspective, the availability of dulaglutide 3.0-mg and 4.5-mg doses may also delay the treatment burden associated with adding or switching medications.

This analysis has limitations that may influence the interpretation of the results. Patients enrolled in the trial were controlled by study-specific criteria; the study included only patients on metformin, with HbA1c levels 7.5% to 11% and with a BMI ≥25 kg/m^2. Therefore, results are not necessarily generalizable to patients with different clinical features (eg, lean patients or patients with HbA1c <7.5%). The analysis included a relatively small number of patients in each of the higher baseline HbA1c subgroups. However, the similar pattern of results in the larger subgroups of patients based on the HEDIS quality measure of HbA1c >9%, and the steeper relationship between baseline HbA1c and change in HbA1c observed with higher doses in the linear regression analysis, both support the findings from the discrete HbA1c subgroups.

In conclusion, these exploratory post hoc analyses reinforce the efficacy of the 1.5-mg dose and provide supportive evidence of the safety and improvement in glycaemic control with dose escalation for patients across a range of baseline HbA1c values. The glycaemic benefit of dulaglutide dose escalation was most prominent in patients with a high HbA1c at baseline. Dulaglutide 3.0 mg and 4.5 mg provide an option to intensify therapy without adding or switching medications, helping clinicians tailor treatment to meet individual patient needs over time.

ACKNOWLEDGMENTS

The AWARD-11 trial was sponsored by Eli Lilly and Company. The authors thank Ciara O'Neill PhD. for writing and editorial support, as well as the trial investigators, trial staff and trial participants for their contributions.

CONFLICT OF INTEREST

Juan P. Frias has received research support from: Allergan, AstraZeneca, Boehringer Ingelheim, BMS, Eli Lilly and Company, Janssen, Madrigal, Merck, Novartis, Novo Nordisk, Pfizer, Sanofi and Theracos, and has served on advisory boards and as a consultant for Altimmune, Axcella Health, Boehringer Ingelheim, Coherus Therapeutics, Eli Lilly and Company, Gilead, Intercept, Merck, Novo Nordisk and Sanofi, and on speaker bureaus for Merck and Sanofi. Enzo Bonora has served on advisory boards for Abbott, AstraZeneca, Becton-Dickinson, Boehringer Ingelheim, Bristol-Myers Squibb, Bruno Farmaceutici, Daiichi-Sanyo, Janssen, Johnson & Johnson, Lilly, MSD, Mundipharma, Novartis, Novo Nordisk, Roche, Sanofi, Servier and Takeda, has received research grants from AstraZeneca, Genzyme, Menarini Diagnostics, Novo Nordisk, Roche Diagnostics and Takeda. Raleigh E. Malik, David A. Cox, M. Angelyn Bethel, Anita Y.M. Kwan and Sohini Raha are current employees and shareholders of Eli Lilly and Company.

AUTHOR CONTRIBUTIONS

David A. Cox, M. Angelyn Bethel, Anita Y.M. Kwan and Raleigh E. Malik contributed to the design and conception of the study. Juan P. Frias, Enzo Bonora and David A. Cox participated in the acquisition of the data. Sohini Raha, David A. Cox, M. Angelyn Bethel, Enzo Bonora, Juan P. Frias, Anita Y.M. Kwan and Raleigh E. Malik contributed to the analysis and interpretation of the data. All authors participated in writing and revising the manuscript.

DATA AVAILABILITY STATEMENT

Lilly provides access to all individual participant data collected during the trial, after anonymization, with the exception of pharmacokinetic or genetic data. Data are available to request 6 months after the indication studied has been approved in the US and EU and after primary publication acceptance. No expiration date of data requests is currently set once they are made available. Access is provided after a proposal has been approved by an independent review committee identified for this purpose and after receipt of a signed data sharing agreement. Data and documents, including the study protocol, statistical analysis plan, clinical study report, blank or annotated case report forms, will be provided in a secure data sharing environment for up to 2 years per proposal. For details on submitting a request, see the instructions provided at vivli.org

ORCID

Juan P. Frias https://orcid.org/0000-0001-9486-1255
David A. Cox https://orcid.org/0000-0003-2052-9836
Raleigh E. Malik https://orcid.org/0000-0002-2797-3545

REFERENCES

1. Garber AJ, Abrahamson MJ, Barzilay JI, et al. Consensus statement by the american association of clinical endocrinologists and american college of endocrinology on the comprehensive type 2 diabetes management algorithm–2016 executive summary. *Endocr Pract*. 2016;22:84-113.
2. American Diabetes Association. Pharmacologic approaches to glycemic treatment: standards of medical care in diabetes—2021. *Diabetes Care*. 2021b;44(Suppl 1):S111-S124.
3. Desouza C, Kirk AR, Mangla KK, Wolden ML, Lingvay I. Real-world clinical outcomes following treatment intensification with GLP-1 RA, OADs or insulin in patients with type 2 diabetes on two oral agents (PATHWAY 2-OADs). *BMJ Open Diabetes Res Care*. 2020;8(2):e001830.
4. Mody R, Huang Q, Yu M, et al. Clinical and economic outcomes among injection-naïve patients with type 2 diabetes initiating dulaglutide compared with basal insulin in a US real-world setting: the DISPEL study. *BMJ Open Diabetes Res Care*. 2019;7(1):e000884.
5. USPI. *Trulicity – dulaglutide injection, solution prescribing information*. USA: Eli Lilly and Company; 2020. https://uspl.lilly.com/trulicity/trulicity.html#pi. Accessed April 1, 2021
6. Frias JP, Bonora E, Nevarez Ruiz L, et al. Efficacy and safety of dulaglutide 3.0 mg and 4.5 mg versus dulaglutide 1.5 mg in metformin-treated patients with type 2 diabetes in a randomized controlled trial (AWARD-11). *Diabetes Care*. 2021;44(3):765-773.

7. National Committee for Quality Assurance. Healthcare Effectiveness Data and Information Set. Comprehensive Diabetes Care. https://www.ncqa.org/hedis/measures/comprehensive-diabetes-care/. Accessed February 22, 2021.
8. American Diabetes Association. 6. Glycemic targets: standards of medical care in diabetes. *Diabetes Care*. 2021;44(Suppl 1):S73-S84.
9. Gallwitz B, Dagogo-Jack S, Thieu V, et al. Effect of once-weekly dulaglutide on glycated haemoglobin (HbA1c) and fasting blood glucose in patient subpopulations by gender, duration of diabetes and baseline HbA1c. *Diabetes Obes Metab*. 2018;20(2):409-418.
10. Gentilella R, Pechtner V, Corcos A, Consoli A. Glucagon-like peptide-1 receptor agonists in type 2 diabetes treatment: are they all the same? *Diabetes Metab Res Rev*. 2019;35(1):e3070.
11. Bihan H, Ng WL, Magliano DJ, Shaw JE. Predictors of efficacy of GLP-1 agonists and DPP-4 inhibitors: a systematic review. *Diabetes Res Clin Pract*. 2016;121:27-34.
12. DeFronzo RA. Insulin resistance, lipotoxicity, type 2 diabetes and atherosclerosis: the missing links. The Claude Bernard lecture 2009. *Diabetologia*. 2010;53(7):1270-1287.
13. Davies MJ, D'Alessio DA, Fradkin J, et al. Management of hyperglycaemia in type 2 diabetes, 2018. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of diabetes (EASD). *Diabetologia*. 2018;61(12):2461-2498.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Frias JP, Bonora E, Cox DA, et al. Glycaemic efficacy of an expanded dose range of dulaglutide according to baseline glycated haemoglobin (HbA1c) subgroup: Post hoc analysis of AWARD-11. *Diabetes Obes Metab*. 2021;23(12):2819-2824. doi:10.1111/dom.14533